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EMG- versus EEG-Triggered Electrical Stimulation for Inducing Corticospinal Plasticity

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***Abstract*—Brain-computer interfaces have been proposed for stroke rehabilitation. Motor cortical activity derived from the electroencephalography (EEG) can trigger external devices that provide congruent sensory feedback. However, many stroke patients regain residual muscle (EMG: electromyography) control due to spontaneous recovery and rehabilitation; therefore, EEG may not be necessary as a control signal. In this study, a direct comparison was made between the induction of corticospinal plasticity using either EEG- or EMG-controlled electrical nerve stimulation. Twenty healthy participants participated in two intervention sessions consisting of EEG- and EMG-controlled electrical stimulation. The sessions consisted of 50 pairings between foot dorsiflexion movements (decoded through either EEG or EMG) and electrical stimulation of the common peroneal nerve. Before, immediately after and 30 minutes after the intervention, 15 motor evoked potentials (MEPs) were elicited in tibialis anterior through transcranial magnetic stimulation. Increased MEPs were observed immediately after (62±26%, 73±27% for EEG- and EMG-triggered electrical stimulation, respectively) and 30 minutes after each of the two interventions (79±26% and 72±27%) compared to the pre-intervention measurement. There was no difference between interventions. Both EEG- and EMG-controlled electrical stimulation can induce corticospinal plasticity which suggests that stroke patients with residual EMG can use that modality instead of EEG to trigger stimulation.**

***Index Terms*—Brain-computer interface, Corticospinal plasticity, Electrical stimulation, myoelectric control, neurorehabilitation.**

I. Introduction

STROKE is one of the leading causes of acquired disability in the world today, with approximately 17 million people suffering a stroke for the first time each year [1]. The consequences of a stroke include cognitive, speech and motor impairments. Approximately 80% of stroke survivors are left with motor impairments [2]. Often these patients are offered some rehabilitation, but more than 50% of them require permanent assistance to perform activities of daily living after rehabilitation has ended [3]-[5]. Because of the heterogeneity of the injury and impairments, there is a multitude of different rehabilitation approaches. However, in general, the effect of these interventions is limited [2].

Interventions recently proposed for the rehabilitation of stroke patients [6], [7] rely on motor learning principles, such as engagement/attention and repetition, and aim to induce plasticity in the brain which is the underlying mechanism for motor learning [8], [9]. Repetition can be obtained through electrical stimulation of nerves and muscles, which has been shown to induce plasticity [10], but patients may be passive during these interventions. By combining electrical stimulation with active movements, so the patient is engaged, the induction of plasticity is enhanced [11]-[13]. This type of intervention can be implemented by asking the participant to execute the movement at specific time instants when the electrical stimulation is delivered [11], [14], [15]. However, movement and stimulation may not be concomitant in time. Alternatively, somatosensory feedback provided by electrical stimulation can be triggered by detecting electromyography (EMG) activation of the affected limb. This type of EMG-triggered somatosensory feedback has been shown to be useful for inducing plasticity or improving motor function in stroke patients [16]-[18]. Movements can also be detected using EEG, where there is little or no residual EMG [19], [20]. This approach provides a brain-computer interface (BCI) which over the recent years, has been shown to induce plasticity and can be used for stroke rehabilitation [21], [22]. The BCI decodes the user's intention to move the affected limb, and in response to the decoded movement, the BCI activates either electrical stimulation [23] or a rehabilitation robot [24], [25] to provide the relevant somatosensory feedback from the affected limb. This approach [23] is also applicable for the rehabilitation of other motor impairment such as spinal cord injury and cerebral palsy.

The major difference between the EMG- and BCI-intervention is the way that the movements are detected. EMG has a much higher signal-to-noise ratio than EEG and therefore it is easier to detect when a muscle is active. For using EMG for movement detection, the patients need to have residual EMG, which is indeed present in most patients [16], [26]. Therefore, it may be easier to use EMG-detected movements to trigger electrical stimulation. However, it is not known which one of the EMG- or EEG-triggered electrical stimulation maximizes the induction of corticospinal plasticity. Using EMG, it is likely to obtain more correct pairings between movement intention and somatosensory feedback from the electrical stimulation

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compared to EEG-triggered electrical stimulation, but with EEG it is possible to detect movement intentions with a latency that is shorter than the EMG; up to 500 ms prior the movement onset [27]-[29]. Thus EEG-triggered electrical stimulation could be better for inducing Hebbian-associative plasticity given a better synchronization between movement intention and somatosensory feedback [30]. Therefore, the aim of this study was to make a direct comparison between the use of EEG and EMG to trigger peripheral nerve electrical stimulation to induce corticospinal plasticity.

II. Methods

A. Participants

Thirty-three healthy participants (15 women: (mean±SD) 27±8 years old) were recruited to participate in the intervention sessions (N=20) and control session (N=20; 7 of these participated in the intervention sessions). All participants gave their written informed consent prior to participation and filled in a questionnaire for transcranial magnetic stimulation (TMS) eligibility based on the recommendations in [31]. All procedures were approved by the local ethical committee of northern Denmark (N-20130081) and carried out according to the Declaration of Helsinki.

B. Experimental Setup

Initially, prior to the experimental sessions, each participant participated in a session where they were familiarized with TMS and electrical stimulation to avoid any uncertainties associated with the first use of these techniques. The experiment was divided into two intervention sessions and one control session; sessions were separated by at least 24 hours. For the individual participant, each session was scheduled for the same time of the day. One intervention consisted of EEG-triggered electrical stimulation, and the other intervention consisted of EMG-triggered electrical stimulation (see Fig. 1). The order of the two intervention sessions was randomized and counterbalanced. The control session was performed after the intervention sessions. Dorsiflexions of the right ankle joint were detected online from continuous EEG or EMG recordings and initiated relevant somatosensory feedback of the deep branch of the common peroneal nerve. The system for detecting the movements based on EEG and EMG was calibrated to the individual participant based on 50 movements that were performed before the interventions started. The intervention lasted until 50 correct pairings between movement and electrical stimulation were obtained. The control session was 2x50 movements without any electrical stimulation applied. The movements in the intervention sessions and control sessions were self-paced. Before the intervention/control, immediately after the intervention/control and 30 minutes after the intervention/control, 15 TMS motor evoked potentials (MEPs) were recorded to quantify corticospinal excitability. During the intervention/control and pre- and post-measurements the participants were instructed to sit as still and relaxed as possible.

C. Movement Intention Detection

EEG: The system for detecting the movements from the continuous EEG has been reported previously [29], and is briefly summarized here. The EEG was recorded from FP1, F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4 (impedance<5 kΩ) according to the international 10-20 system using sintered Ag/AgCl ring electrodes with a sampling frequency of 500 Hz (EEG amplifiers, Nuamps Express, Neuroscan). The reference and ground electrodes were placed on the right mastoid and on the forehead, respectively. The signals were bandpass filtered from 0.05-10 Hz with a 2nd order zero-phase shift Butterworth filter and spatially filtered around Cz. FP1 was used to monitor electrooculography (EOG). The system used template matching to identify the movements, i.e. when the output of the matched filter exceeded a given threshold a movement was detected. The template was obtained from the 50 movements for the calibration. The filtered EEG signals were averaged across the 50 movements, and the initial negative phase of the movement-related cortical potential was extracted which was defined as the data from the EMG onset and 2 s prior this point [19]. Based on cross-validation in the training data set, a receiver operating characteristics curve was obtained from which the detection threshold was selected to obtain a trade-off between the true positive rate (TPR) and the number of false positives per minute (FPs/min). The system was deactivated when the EOG activity in FP1 exceeded a threshold, which was selected for each participant. The template, detection threshold and EOG threshold were used in the online intervention. The system imported data every 100 ms. When a movement was detected, electrical stimulation was delivered. To avoid multiple stimuli occurring immediately after each other, the detector was disabled for 5 seconds. The TPR, FPS/min, and time taken to complete the task (T_t) were recorded to evaluate the system performance. The TPR was calculated as the number of correctly detected movements divided by the total number of movements performed. These metrics were also obtained for the EMG detector.

Surface EMG: Bipolar EMG was recorded using two surface EMG electrodes (20 mm Blue Sensor Ag/AgCl, AMBU A/S, Denmark) placed on the right tibialis anterior muscle. The ground electrode was placed on the tibia. The EMG was sampled with the same amplifier as for the EEG with the same sampling frequency. The signal was bandpass filtered from 10-200 Hz with a 2nd order zero-phase shift Butterworth filter. Moreover, the signal was notch filtered from 49 to 51 Hz. The filtered signal was then rectified. The maximum amplitude of the rectified EMG during the movements was identified and then 10% of this value was calculated and used as the threshold that was used in the online system [29]. The detector was

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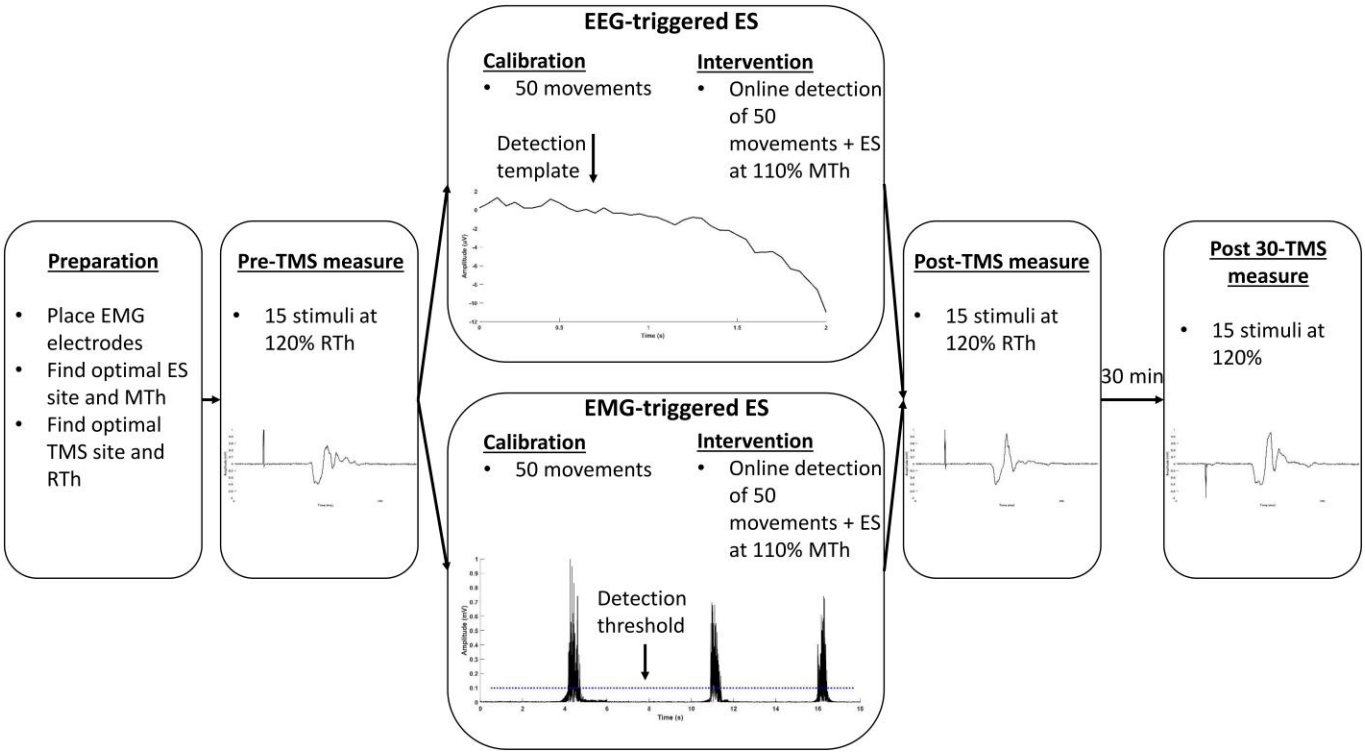

Fig. 1. Schematic overview of the experimental sessions. In the TMS measurements MEPs are shown for a representative participant from the same session (EEG). Moreover, the EEG template for the movement intention and the EMG during three movements are shown. Abbreviations: "ES": Electrical stimulation, "MTh": Motor threshold, and "RTh": Resting threshold.

disabled for 5 seconds after a movement was detected.

D. TMS and MEPs

MEPs were elicited in tibialis anterior with single pulse TMS using a Magstim 200 (Magstim Company, Dyfed, UK) and a figure-of-eight double cone-coil with a posterior-anterior current direction. Prior to the pre-TMS measurement (15 MEPs before the intervention/control), the optimal stimulation site was determined. It was defined as the area where the largest peak-peak amplitude MEPs were elicited compared to the adjacent areas. The position of the coil was marked on the participant with a marker to ensure that the coil was placed at the same position throughout the experimental session. Afterwards, the resting threshold was determined which was defined as the lowest intensity needed to elicit 5 out of 10 visible MEPs (peak-peak amplitudes larger than ~50 μV). In the pre-, post- and post-30 min measurements, 15 stimuli were delivered at 120% of the resting threshold; each stimulus was separated by 5-7 seconds. The MEPs were recorded using the same electrodes as in the EMG intervention, but they were amplified with a customized amplifier (Jan Stavnshøj, Aalborg University) with a gain of 1000. The signals were sampled at 4000 Hz using the Mr. Kick software (Knud Larsen, Aalborg University).

E. Electrical Stimulation

The electrical stimulation (STMISOLA Linear Isolated Stimulator, BIOPAC Systems, Inc.) was applied to the deep branch of the right common peroneal nerve, which innervates tibialis anterior, using two stimulation electrodes (32 mm, PALS, Platinum, Patented Conductive Neurostimulation Electrodes, Axelgaard Manufacturing Co., Ltd., USA). The stimulation electrodes were placed over the skin of the nerve. The proximal electrode was the cathode and the distal electrode was the anode. The optimal stimulation site was found by placing the stimulation electrodes that evoked activity in tibialis anterior only without eliciting activity in synergistic or antagonistic muscles; this was identified through palpation of the muscles. Next, the motor threshold was found, which was the lowest intensity needed to evoke a palpable response in the tibialis anterior tendon. This was determined before the pre-TMS measurement. In the intervention, a 1 ms wide biphasic square pulse with an intensity of 110% of the motor threshold was delivered.

F. Statistics

The statistical analysis was performed in R (R Foundation for Statistical Computing) version 3.5.1 using *lme4* package version 1.1-21 [32], [33], *robustlmm* package version 2.3 [34] and *emmeans* package version 1.3.4 [35]. Tukey's HSD method was used to perform pair wise contrasts. Statistical significance was considered below 0.05. The detailed statistical analysis reports are given in the supplementary files. Linear mixed regression models were setup to investigate the following: (a) The pre- to post-/post-30 effect of EEG and EMG based interventions in comparison with control and to each other. (b) The influence of the detection performance of EEG and EMG based BCIs on their effect sizes.

We investigated the treatment effect in terms of the induced plasticity measured as the average peak-peak MEP amplitude. The average was obtained from 15 trials. The absolute

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measurements (mV) and the relative measurements (% change) were used for analysis. The subject wise % change was calculated as (post – pre) / pre × 100.

The following linear mixed regression models were setup to investigate (a) in terms of absolute units and relative units respectively. The models are presented as R formulas [32].

$$\mathrm{MEP_{abs}} \sim \mathrm{MEP_{pre}} + \mathrm{Session} \times \mathrm{Time} + (1 \,|\, \mathrm{Subject}) \quad (1)$$

$$\mathrm{MEP_{\%}} \sim \mathrm{MEP_{pre}} + \mathrm{Session} \times \mathrm{Time} + (1 \,|\, \mathrm{Subject}) \quad (2)$$

$\mathrm{MEP_{abs}}$, $\mathrm{MEP_{\%}}$ and $\mathrm{MEP_{pre}}$ were codified as continuous variables. Session, Time and Subject were entered as categorical variables (see Table I and II). The models estimated MEP measurements (mV, % change) for the three sessions (EEG, EMG, Control) at the two time points (post-, post-30). Moreover, the models controlled for the baseline values and estimated between subject variance to fit the repeated measures design of this study. These models were based on model 1 proposed by Twisk et. al. for evaluating treatment effects in randomised controlled trials [36]. For model (1), Gamma distribution and identity link were used since the peak-peak MEP amplitudes were always positive and were distributed with a positive skew. In model (2), Gaussian distribution and identity link were used. Model (2) was fitted using robust linear regression to avoid unwarranted influence of outliers [34]. Further details are given in the supplementary files.

We investigated the relationship between the detection performance of the BCIs and the treatment effects using similar linear mixed regression models. This was done only in terms of the absolute units (mV). FPs/min, T_t, and the TPR were used as the measure of the detection performance of the detectors (based on EMG and EEG). The analysis consisted of two stages: (i) a blind covariate evaluation as proposed by Kunz et. al. [37], and (ii) significance testing on the model finalised using the blind covariate evaluation. The purpose of blind covariate evaluation was to minimise bias and to eliminate unnecessary covariates which can add noise to the model. The following model was used for stage (i).

$$\mathrm{MEP}_{abs} \sim \mathrm{MEP_{pre}} + \mathrm{Time} + \mathrm{TPR} + \mathrm{FPs/min} + \mathrm{T_t} + (1|\mathrm{Subject}) \quad (3)$$

TPR, FPs/min and T_t were entered as continuous variables. Gaussian distribution and identity link were used for this model. A notable feature of this model is the missing Session variable which was not entered for the sake of blinding. Data corresponding to the EEG and EMG sessions was used to setup this model. Data from control session was not used as it did not have the performance metrices. Variance explained by each covariate was obtained as semi-partial R^2 statistic [38]. Any covariate which explained less than 5% variance was eliminated from further analysis. Finally, for (ii), following model was setup.

$$\mathrm{MEP}_{abs} \sim \mathrm{MEP_{pre}} + \mathrm{Session} \times \mathrm{Time} + \mathrm{Session} \times \mathrm{TPR} + (1 \,|\, \mathrm{Subject}) \quad (4)$$

This model estimated separate linear trends between TPR and $\mathrm{MEP_{abs}}$ for sessions corresponding to EEG and EMG. Gamma distribution and identity link were used for this model.

TABLE I
PRE- TO POST- EFFECT SIZES FOR MEP AMPLITUDES AND CHANGES ESTIMATED FROM THE STATISTICAL MODELS AT $\mathrm{MEP_{PRE}} = 0$

Time	Session	$\mathrm{MEP_{abs}}$ ±SE (mV)	95% CIs (mV)	z, p, H_0: μ=0
Post	EEG	0.38±0.17	0.04, 0.72	z=2.2 **p=0.03**
	EMG	0.56±0.18	0.20, 0.92	z=3.05 **p=0.002**
	Control	0.24±0.15	-0.06, 0.54	z=1.6, p=0.12
Post-30	EEG	0.53±0.19	0.17, 0.89	z=2.85, **p=0.004**
	EMG	0.53±0.19	0.17, 0.90	z=2.87, **p=0.004**
	Control	0.24±0.15	-0.06, 0.54	z=1.59, p=0.11

Time	Session	$\mathrm{MEP_{\%}}$ ±SE (%)	95% CIs (%)	z, p, H_0: μ=0
Post	EEG	62.1±25.7	11.8, 113	z=2.42 **p=0.02**
	EMG	73.3±27.0	20.4, 126	z=2.71 **p=0.007**
	Control	30.2±24.1	-17, 77.4	z=1.25 p=0.21
Post-30	EEG	79.2±25.7	28.9, 130	z=3.08 **p=0.002**
	EMG	72.3±27.0	19.5, 125	z=2.68 **p=0.007**
	Control	39±24.1	-8.1, 86.2	z=1.62 p=0.15

Significant effects ($p<0.05$) are in bold text.

TABLE II
CONTRASTS FOR MEP AMPLITUDES AND CHANGES FROM THE STATISTICAL MODELS AT $\mathrm{MEP_{PRE}}$=0

Time	Contrast	Difference ±SE (mV)	z, p, H_0: μ=0
Post	EEG-EMG	-0.18±0.11	z=-1.6 p=0.25
	EEG-Control	0.14±0.10	z=1.38 p=0.35
	EMG-Control	0.32±0.11	z=2.87 **p=0.01**
Post-30	EEG-EMG	-0.004±0.13	z=-0.30 p=0.99
	EEG-Control	0.29±0.13	z=2.28, p=0.06
	EMG-Control	0.29±0.12	z=2.43, **p=0.04**

Time	Contrasts	Difference ±SE (%)	z, p, H_0: μ=0
Post	EEG-EMG	-11.1±33.1	z=-0.34 p=0.94
	EEG-Control	31.9±33.3	z=0.96 p=0.60
	EMG-Control	43.1±33.8	z=1.27 p=0.41
Post-30	EEG-EMG	6.9±33.1	z=0.21 p=0.98
	EEG-Control	40.29±33.3	z=1.21 p=0.45
	EMG-Control	33.3±33.8	z=0.98, p=0.59

Significant effects ($p<0.05$) are in bold text.

The z-statistics are presented as well as the p-value, which is considered significant if $p<0.05$.

III. RESULTS

A. MEP Size

The peak-peak MEP amplitudes of individual subjects are plotted in Fig. 2. The individual trends indicate a larger increase in pre- to post-MEP amplitude for EEG and EMG sessions with respect to the control session.

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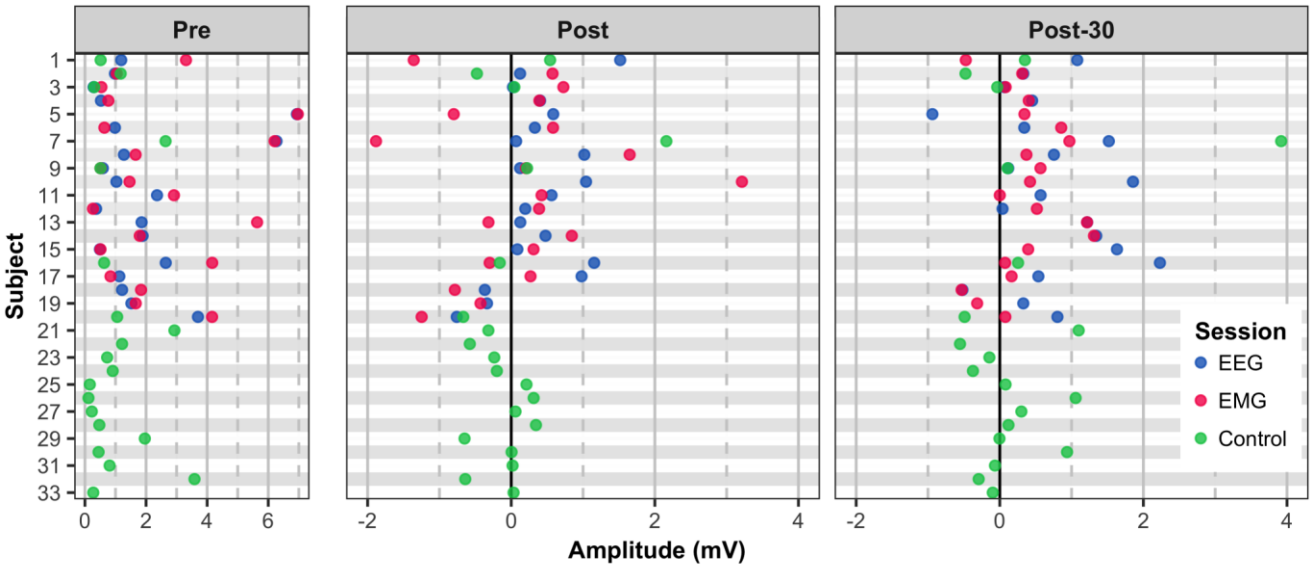

Fig. 2. Peak-peak MEP amplitudes. The left graph shows the raw baseline values (Pre). The other graphs show the adjusted post and post 30 MEPs with respect to the baseline set to 0 (black vertical line). The individual trends indicate a larger increase in pre- to post-MEP amplitude for EEG and EMG sessions with respect to control session.

TABLE IV
CONTRASTS FROM THE STATISTICAL MODEL AT TPR=85.40% AND MEP_{PRE} = 0

Time	Contrast	Difference ±SE (mV)	z, p, H_0: μ=0
Post	EEG-EMG	-0.42±0.22	z=-1.93 p=0.054
Post-30	EEG-EMG	-0.29±0.22	z=-1.30 p=0.20

TABLE III
PRE- TO POST- EFFECT SIZES FROM THE STATISTICAL MODEL AT TPR=85.40% AND MEP_{PRE} = 0

Time	Session	MEP_{abs} ±SE (mV)	95% CIs (mV)	z, p, H_0: μ=0
Post	EEG	0.65±0.25	0.16, 0.14	z=2.59 **p=0.01**
	EMG	1.07±0.33	0.43, 1.17	z=3.28 **p=0.001**
Post-30	EEG	0.75±0.25	0.25, 1.25	z=2.97 **p=0.003**
	EMG	1.04±0.33	0.40, 1.68	z=3.20 **p=0.001**

Significant effects (p<0.05) are in bold text.

TABLE V
TREND BETWEEN TPR AND MEP_{ABS} FROM THE STATISTICAL MODEL WHICH CONTROLLED FOR THE TPR

Session	Trend ±SE (mV/TPR-ratio)	z, p, H_0: μ=0
EEG	-0.58±0.52	z=-1.14 p=0.25
EMG	-1.67±1.86	z=-0.91 p=0.36

MEP Size – Absolute Units: The pre- to post-effect sizes for MEP absolute amplitude estimated from the statistical model (1) are given in Table I (top). Table II (top) shows pair-wise contrasts across sessions at the two time points (post- and post-30). MEP amplitude was significantly different between post-EMG and post-Control sessions as well as post-30.

MEP Size – Relative Units: The pre- to post-effect size of MEPs percentage change estimated from the statistical model is given in Table I (bottom). Table II (bottom) shows pair-wise contrasts across sessions at the two time points (post- and post-30). MEPs percentage change was not significantly different among sessions at post- and post-30 time points.

These results indicate a statistically significant ($p < 0.05$) increase in MEP amplitudes (on both absolute and relative scale) from pre- to post- and post-30 for EEG and EMG paradigms. The effect sizes for EEG and EMG were consistently larger than the Control. Although not statistically significant ($p > 0.05$), the effect of EMG was larger than EEG at post-, whereas the difference between the two at post-30 was very small.

B. *Effect of System Performance*

The mean TPR was 85.40 ± [41 100] % ([min max]) and explained 7.6% of the variance in MEP amplitudes. The mean FPs/min was 0.52 ± [0 2.4] and explained 0.1% of the variance. Lastly, the mean T_t was 11.35 ± [6 33] min and it explained 3.1% of the variance in MEP amplitudes. Since FPs/min and T_t explained less than 5% of the variance they were removed from further analysis. The estimated effect sizes for EEG and EMG from the statistical model (4) which controlled for the TPR (mean value = 85.40%) are given in Table III and Table IV. These results suggest that when controlled for TPR, the effect sizes for both EEG and EMG and their contrasts were larger.

The estimated trends for TPR obtained from the statistical model are given in Table V. There was a negative association between TPR and MEP amplitude. These trends were, however, not statistically significant.

IV. DISCUSSION

The aim of this study was to investigate if there was a difference between EEG- and EMG-triggered electrical stimulation on the induction of corticospinal plasticity. It was shown that both methods could induce corticospinal plasticity and their effect was larger than the control. Although not statistically significant, EMG had a larger effect at post- compared to EEG when the effect of TPR was not statistically controlled. The effect of EMG was larger yet statistically not

significant at both post- and post30- when the effect of TPR was statistically controlled. This was most likely due to the lower detection performance of EEG compared to EMG in this study. Finally, there was a negative association between TPR and change in MEP amplitude for both EEG and EMG.

A. *Induction of Plasticity*

The findings from previous studies regarding induction of corticospinal plasticity have been validated, and the induction of plasticity is in the range of what has been reported previously [11], [12], [14], [23], [24], [39]. There was a considerable amount of variability among the participants indicated by the standard error, which could be due to various factors such as attention or anatomical differences of the excitability of the neurons in the motor cortical representation of the foot as well as how comfortable the participants were with the TMS [40]. The MEP size increased for most participants from the pre-measurement to the post-measurements. The two intervention sessions were different from the control session (only EMG-triggered electrical stimulation was significant) although movement alone also increased the MEP size, which is an agreement with previous findings [11]. Providing single-pulse peripheral electrical stimulation alone with the same number of electrical stimuli as in this study has previously been shown not to induce inducing corticospinal plasticity [12], [14], which highlights the need for a temporal association between motor cortical activity and somatosensory feedback. The difference between movement alone and movement paired with electrical stimulation could be due to extra afferent inflow from the electrical stimulation. The findings in this study also suggest that the strict temporal association between the motor cortical activity and the inflow of the somatosensory feedback can be obtained using EMG, which means that movement prediction from EEG may not be necessary. As has been outlined previously in similar studies [14], [24], the proposed mechanism for the changes in corticospinal plasticity could be long-term potentiation-like plasticity due to different criteria: rapid onset, lasting effects, associativity and specificity. The post-measurement indicated a rapid onset which was persistent after the intervention ended, and the post-30 min measurement indicated that the changes were long-lasting. It was shown previously that this associative intervention is specific as well [14]. A limitation of this study is that it is not known where in the corticospinal pathway the changes occur. It has been suggested using a similar protocol that the changes are cortical/supraspinal [23] based on recordings of stretch reflexes; in that study motor imagination was used instead of motor execution as in the current study. Motor execution modulates the spinal excitability, so it could be hypothesized that some of the changes in corticospinal excitability could be due to spinal excitability. This should be validated in future studies where stretch reflexes and/or F-waves are elicited before and after the interventions. Additional measurements could be performed to determine the origin of the changes; these could include functional near-infrared spectroscopy and connectivity analysis of the brain [41].

B. *System Performance*

The detection performance of the system was as expected much better when using the EMG. The performance of the system based on EEG was comparable to previous studies [23], [42], [43], but given a much higher signal-to-noise ratio of the EMG, it is natural that the performance was higher. A limitation of this study, however, was that the participants were healthy subjects on the contrary to the intended end-users which will be neurological patients with motor paresis or paralysis. Obviously, if there is no residual EMG or much spasticity, then EMG will not be ideal for detecting the attempted movements. It has been reported that it is difficult to decode complex and precise movements from stroke patients using EMG [44], but the opposite has also been shown [45], which may indicate that the amount of residual EMG activity determine the performance of the movement detection system. The number of movement types (e.g. grasp types) that needs to be decoded can also be very limited (one movement in this study), which will improve the system performance.

C. *System Performance and Plasticity Induction Interaction*

Currently in the literature, the relationship between the algorithmic performance of a BCI system and its efficacy in inducing plasticity is unknown [30]. Niazi et al. (2012) showed a significant correlation in induced plasticity and BCI performance but this is related to a small sample size (N=8). In this study, the slopes between TPR (both session EEG and EMG) and MEP size were negative, but there was not enough evidence to suggest that the slopes are non-zeros. This is counter intuitive and requires further investigation. In this study, TPR was an observed variable and was used to statistically control for any differences across sessions. Thus, its negative association with the outcome variable (MEP size) cannot be concluded as a causal effect. A possible explanation could be that more movements are performed when the TPR is low, and the extra movements that are performed lead to an increase in MEP size. In a future study, the causal relationship between TPR and MEP size can be studied by deliberately maintaining two or three levels of TPR across groups while controlling for all the other variables. This controlling of TPR might be easier to achieve for EMG.

D. *Practical Aspects*

In recent years, several studies have suggested and evaluated the use of BCIs to induce plasticity for stroke rehabilitation [15], [22], [46]-[50], but based on the findings in the current study, it may not be needed to use BCIs given the patients have residual EMG. In a recent study, it was also suggested to use BCI for stroke rehabilitation by training the patients to obtain discriminable EMG activity in the target muscles, and then switch to EMG detection afterwards that is more reliable [16]. Thus, EEG may be necessary only in the acute and sub-acute phases when there is no detectable EMG, and then it could be substituted by EMG. In the transition from EEG to EMG, signals from peripheral nerves could be used as well [51].

V. Conclusion

The EEG- and EMG-triggered electrical stimulation can be used to induce corticospinal plasticity, and there was no difference in the amount of plasticity that was induced using the two modalities. The movement detection was much higher using EMG compared to EEG. Thus, in motor rehabilitation after neurological injury, it is suggested to trigger electrical stimulation using EMG if it is detectable. However, the findings in the current study should be validated in a randomized controlled trial with the end-users, patients with neurological motor disorders.

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